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THE DEVELOPMENT OF A NEW BLOOD SUPPLY TO THE HEART BY OPERATION*

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Two relatively small arteries supply the most vital muscular structure of the body. This muscular organ is in constant motion, and to make its movements free and frictionless it is enclosed in a moist envelope of mesothelium. In providing man with this anatomic pattern Nature so frequently has deprived him of an important compensatory mechanism, namely, the ability to develop an adequate collateral blood supply to this organ. The heart is anchored in the body by the great vessels at its base, and while the entire surface of the heart is in direct contact with adjacent structures it has a minimum of direct continuity with the rest of the body. The only continuity that the heart has with the rest of the body is through the walls of the great vessels, some fat, a few nerves and lymphatics that constitute its anchorage, and these structures are relatively avascular tissues. This anatomic arrangement has made the heart a defenseless organ when its normal blood supply is interrupted. The appalling incidence of sudden death from heart failure attests to the destructive nature of coronary closure. Under normal conditions the myocardium receives practically all its blood through the left and right coronary arteries. The thebesian channels opening directly from the left ventricular cavity offer a second possible source of blood supply.¹ The importance of these channels has been the subject of argument. It has been claimed by some that the flow through the thebesian vessels is of little importance. This statement is based on the assumption that the thebesian channels are compressed, perhaps completely collapsed, by the tension of contracting muscle fibers during systole when the pressure in the ventricular cavity is being built up, and as these channels open during diastole the

* Some of the experimental data were presented in an address to the Caduceus Honorary Society of the Emory University Medical School on May 17, 1934.

pressure in the ventricular cavity synchronously falls.¹ The third possible source of blood supply to the heart is through the extracardiac anastomoses that are present in the tissues at the base of the heart.² When the blood flow through the coronary arteries is reduced gradually, compensatory mechanisms are established. Complete closure of a major coronary artery is compatible with life, if the closure takes place sufficiently slowly. Indeed, complete closure of both major coronary arteries may be compatible with life,³ but the number of times that the process of occlusion has gone on to completion in each of the two major coronary arteries is excessively small. The occlusion must occur at such a rate as to permit the other sources of blood supply to develop. Usually while this substitution is being made, life is snuffed out like a candle flame.

I have been interested in the heart as a surgical organ since 1923, and during this period of time a rather extensive background of surgical experimentation on the heart has been accumulated, as the result of over 1200 experimental operations which have been done by my assistants and myself. I made the observation several years ago that blood vessels extended between the heart and an adherent scar. At that time Dr. R. A. Griswold⁴ and I were interested in studying chronic compression of the heart produced experimentally and we were trying to resect an adherent scar from the surface of the heart. In so far as these small vascular connections between the heart and adherent tissues were concerned, we simply noted their presence. At that time they had no other significance for us. Last November, while a compressing scar was being resected from a human heart, a broad band of scar extending from the base of the left ventricle to the parietal pericardium was transected. Brisk bleeding occurred from the cut ends of the scar and the bleeding was more brisk from the cardiac end than from the pericardial end. This was the first direct observation made at the operating table that blood actually flowed between myocardium and adherent tissues. This observation had now assumed new and additional significance because in the meantime we had been attempting to produce a collateral vascular bed to the heart experimentally. This observation confirmed our belief that vascular connections between heart and adherent tissues could be produced in the human being. I desire to give credit to Dr. Alan R. Moritz for directing my attention to the subject of vascularization of cardiopericardial adhesions. Moritz, Hudson and Orgain⁵ not only demonstrated anatomically the presence of blood vessels in cardiac adhesions by the injection of carbon particles into the coronary arteries but also Doctor Moritz believed that under certain conditions these blood vessels might function and become an important source of blood supply to the heart. He directed my attention to a case reported by Thorel in 1903. This patient had complete obliteration of both major coronary arteries, and Thorel suggested the possibility of the heart receiving a supply of blood through adhesions that were present.

Could the heart be given a new source of blood supply by operation? I carried out the first experiments in an attempt to study this problem in

February, 1932, and shortly thereafter Dr. V. L. Tichy collaborated with me, not only in the technical surgery that was involved but also in the solution of many problems.⁶ Doctor Moritz followed the work with interest and gave us valuable suggestions concerning the injection and study of the specimens.^{7, 8} In these experiments the collateral vascular bed consisted of parietal pericardium and pericardial fat. The epicardium and the lining of the parietal pericardium were removed with a burr because it was our belief that these structures acted as a barrier to the growth of blood vessels into the heart. The results of these experiments were as follows: (1) Almost total occlusion of right and left coronary arteries near the aorta was compatible with life if the heart had been provided with a collateral vascular bed. The occlusion of arteries was produced by silver bands which were compressed in stages at repeated operation. (2) Dye penetrated the myocardium through the collateral bed, and we assumed that if particles of dye entered the myocardium as freely as they did, that blood also flowed into the myocardium through these vascular channels. We believe that we succeeded in giving the heart a new source of blood supply and that this was sufficient to maintain cardiac function. (3) A pressure-differential was necessary to promote anastomosis between the cardiac and extracardiac vascular beds. In other words, a physiologic need for blood in the myocardium was necessary for such anastomoses to develop. The physiologic need for blood was produced by occlusion of the major coronary arteries by silver bands which were placed around the arteries and which were compressed in stages at successive operations. (4) The development of anastomoses between the myocardium and the collateral bed was demonstrable three weeks after operation. More recently we have succeeded in demonstrating anastomoses between extracardiac and cardiac beds two weeks after the bed was applied to the myocardium.

During the past eight months additional experimental operations have been carried out with the assistance of Dr. Ernest Bright and Miss Alice B. Maltby. In these experiments pedicle grafts of muscle were used together with the pericardial and mediastinal fat for the vascular bed. Omentum was also brought up through an opening in the diaphragm and sutured to the heart. These experiments have not yet been published but I shall refer briefly to some of the results:

(1) Anastomoses readily develop between skeletal muscle and cardiac muscle provided the normal blood supply to the heart has been reduced. If the coronary arteries were not partially occluded the anastomoses between omentum or skeletal muscle and myocardium, although present to some extent, did not become well developed.

(2) A collateral vascular bed gives the heart partial, but not complete, protection when the right coronary artery is occluded in one stage. When the descending ramus of the left coronary artery is ligated in one stage, the degree of protection afforded by a collateral vascular bed is slight. The conclusion that can be drawn is that a collateral vascular bed protects the heart when the right coronary artery is occluded in one stage. The larger

the coronary artery occluded, the less is the protective effect. The evidence, however, was definite that the presence of a collateral vascular bed protected the heart from the ravages of sudden occlusion of a major coronary artery. In this respect the operation becomes a prophylactic measure, and if the experimental data can be applied to patients with coronary sclerosis it would seem that this operation should be done early in the course of the disease, before replacement of myocardium by scar tissue and fat has taken place or before the heart has been brought to a standstill by occlusion.

(3) The right coronary artery, the ramus descendens of the left coronary artery, or the ramus circumflexus of the left coronary artery, can be ligated successfully almost as a routine if the ligation is carried out in two stages.

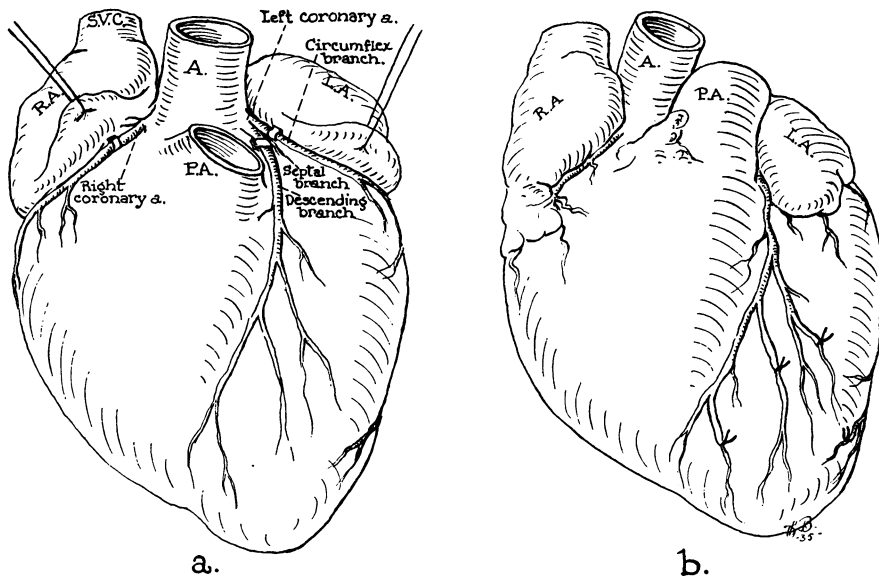


FIG. 1.—Two types of operations are shown: (a) shows the usual sites where silver bands were applied to the coronary arteries. A reduction of 30 to 50 per cent in the cross sectional area of these three arteries was compatible with life. (b) shows another type of experiment. Four or five peripheral branches of the coronary arteries over the apex of the heart were ligated. This experiment was always fatal. In (a) the reduction in total coronary blood flow was considerably greater than in (b). The heart cannot tolerate ischemia of a severe degree even though the area of ischemia is small. The collateral bed produced by operation can transport blood to such ischemic areas.

The explanation for this is that the reduction in blood flow brought about by the partial constriction of this vessel—and as little as 20 per cent of the cross section is efficacious—is compensated for by the development of collateral vessels. After this compensatory mechanism has been established, complete occlusion of the artery does not produce complete ischemia of the muscle. The myocardium remains viable after complete occlusion and additional channels become established.

(4) Distribution of blood to every part of the myocardium is of vital importance. If one relatively small portion of the myocardium is rendered ischemic by the peripheral ligation of four or five arterial branches, ventricular fibrillation develops and this is routinely fatal (Fig. 1). An equal

distribution of blood to the myocardium is essential for maintenance of function.

(5) The collateral vascular bed is functional not only in making a new source of blood supply available to the heart but it also helps to distribute blood to various portions of the myocardium. In this respect the collateral vascular bed produced by operation acts as an anastomotic bridge that transports blood from the bed of one coronary vessel to the bed of another coronary vessel where the blood flow is deficient.

The question naturally arises as to whether or not the presence of the new vascular bed might interfere with the movement of the heart. The importance of adhesions to the heart has been greatly overemphasized in the past. Cardiopericardial adhesions usually are silent lesions of little or no clinical significance. In none of our experiments did we find that the circulation was in any way embarrassed by these adhesions. Adhesions to the heart can embarrass the circulation in several ways: (1) A constricting bed of scar tissue may produce chronic cardiac compression. Adhesions in this clinical syndrome are entirely incidental and of no significance. (2) Adhesions between the heart and chest wall may act as a harness through which the heart pulls and expends energy. In our experiments the heart was not so extensively and intimately bound to the chest wall as to produce embarrassment. (3) The heart may be sharply angulated from its normal axis by such adhesions so that it cannot effectively function.

With this experimental work as a background it was decided to attempt to apply the data to a patient suffering from coronary sclerosis. To select a satisfactory case was not without difficulty and then to have the selected patient give his consent to have an operation performed on his heart (an operation that had never been done before on a human being) required something of the heroic spirit. For this reason I wish to mention the name of the patient, Joseph Krchmar, of Chardon, Ohio. I believe he has made a contribution to surgery. I also want to give credit to his physician, Dr. Walter Corey of Chardon. Doctor Corey had the imagination to see the possibilities of the experiments and he made a serious attempt to secure a patient for the operation. I also want to thank the internists, Dr. Joseph T. Wearn and Dr. Harold Feil. Doctor Feil is collaborating in the clinical aspects of this work.

CASE REPORT

J. K. was admitted to the Lakeside Hospital February 3, 1935. He was 48 years old, a white male, married, formerly a coal miner, more recently a farmer. His complaint was pain in the chest, over the heart, to the left of the sternum. He remembers the onset of the pain distinctly. A sharp pain appeared suddenly over the heart while he was at work nine years ago. It was accompanied by dyspnea and dizziness. After this initial attack he went back to fairly heavy labor until five years ago. Then, because of repeated attacks of substernal oppression, he moved to a farm. The precordial oppression was accentuated after meals. The patient claims that he had not done heavy work for the past five years, but he has done such things as plowing. During the last year or two he suffered from attacks of sharp, knife-like pains over the heart. These radiated to the left shoulder and down the left arm to the elbow. During these attacks of pain

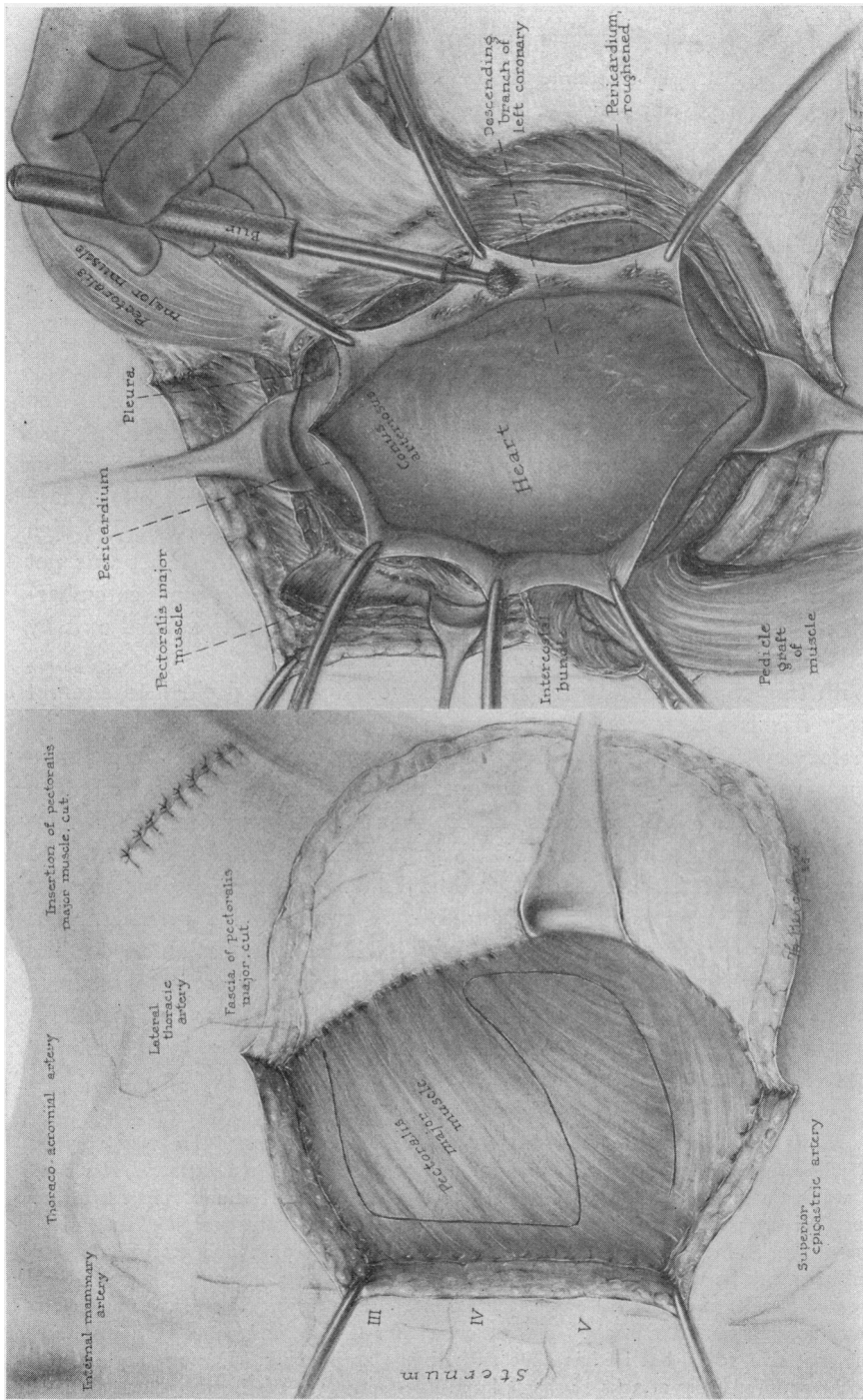


FIG. 2.—The insertion of the pectoralis major muscle was incised. A flap of skin and pectoral fascia was turned laterally. The pectoral muscle was incised as shown. This provided a pedicle graft of muscle with its attachment lateral to the sternum.

FIG. 3.—The pericardium was opened. The inner surface of the pericardium is roughened with a burr.

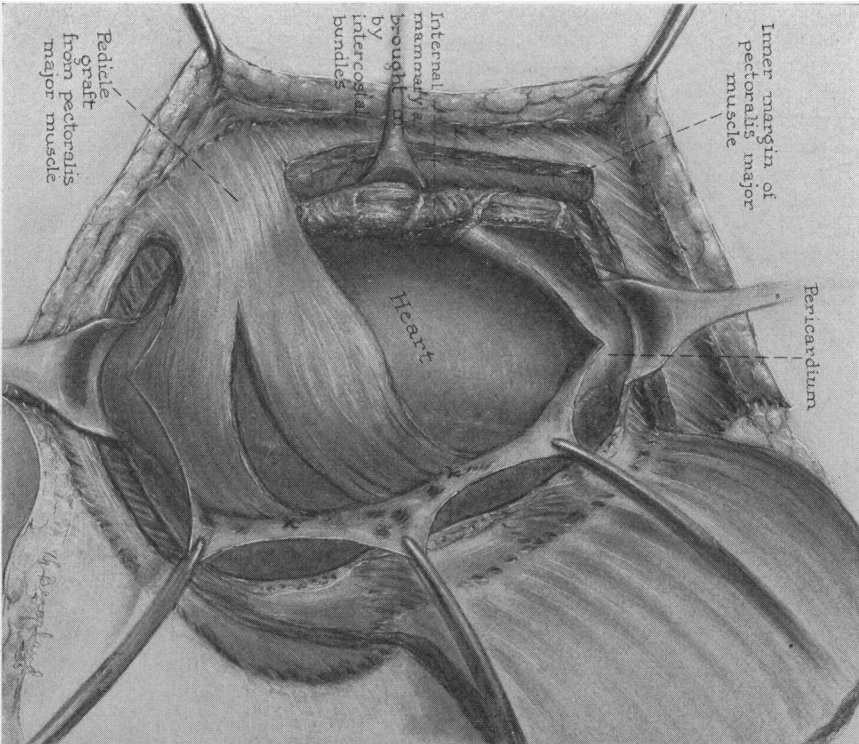


FIG. 4.—The pedicle grafts were carried posteriorly to the circumflex bed and sutured to the parietal pericardium. The intercostal bundles were carried beneath the sternum and sutured to the parietal pericardium. This carried the internal mammary vessels onto the surface of the heart.

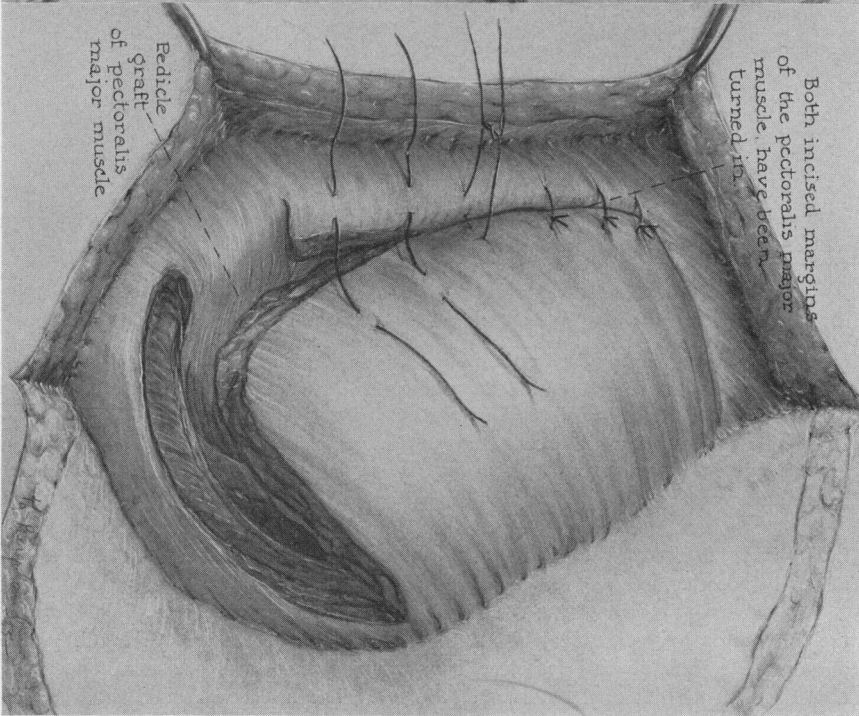


FIG. 5.—The parietal pericardium was left open and the incised margins of the pectoral muscles were inverted so that they came into contact with the heart.

he felt suffocated, was dyspneic and the heart palpitated. Epigastric distress after meals was also a prominent symptom. Numbness and tingling of the left hand and fingers frequently accompanied the pain. Ten days before admission to the hospital the patient, while carrying some wood into his house, was seized with violent, sharp, precordial pain, dyspnea and dizziness. He went to bed where he remained until he came to the hospital.

The patient was well developed, well nourished and of a phlegmatic temperament. His expression was worried. Examination of the precordium was negative. The heart was slightly enlarged to the left. On admission to the hospital the systolic pressure was 155 and the diastolic pressure was 95 Mm. Hg. The peripheral arteries were moderately thickened and tortuous. Exercise produced substernal oppression but no sharp pain. The electrocardiogram after exercise showed no significant change. While in the hospital the

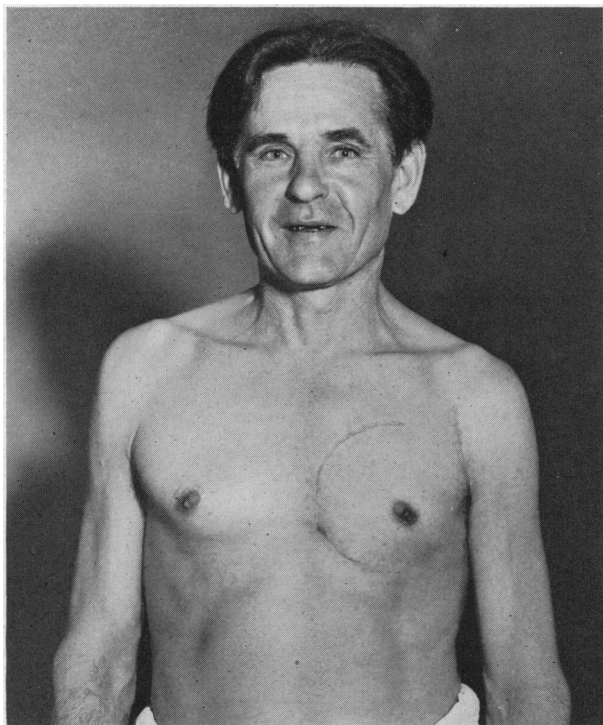


FIG. 6.—Patient three and one-half months after operation.

patient suffered a sudden, severe, knife-like attack of pain after taking a bath. The pain was precordial, to the left of the sternum, and did not radiate. At the same time he became cyanotic, very dyspneic and extremely apprehensive. Amyl nitrite gave him relief. A diagnosis of coronary sclerosis and angina pectoris was made. The electrocardiographic findings would indicate that there was no bundle damage thus far. He also had generalized arteriosclerosis and a mild degree of hypertension.

The patient consented to operation. He desired to go home to visit with his family but he returned to the hospital at midnight because of pain and a fear of impending death.

Operation.—February 13, 1935. Gas-oxygen anesthesia was used. The insertion of the left pectoral muscle was incised at the suggestion of Dr. W. C. McCally, for the purpose of mobilizing this muscle. This wound was closed. A curved incision was then made to the left of the sternum. (Fig. 2). The fascia was dissected from the left

pectoral muscle. The inferior portion of the pectoral muscle was incised for the purpose of making a pedicle graft. An incision was then made through the pectoral muscle parallel to the sternum exposing the third, fourth, and fifth costal cartilages. The muscle was freed from the chest wall. These cartilages were removed. The intercostal bundles were cut laterally leaving them attached to the region of the internal mammary vessels. The pericardium was incised from base to apex (Fig. 3). The lining of the pericardium everywhere was roughened by means of a burr. The epicardium was removed in shreds by means of a burr. This produced a great many extra systoles and some dilatation of the heart. Rest periods were given. At the suggestion of Doctor Wearn I tried to palpate the coronary vessels but with the heart moving I could not be sure that I could feel them. The pedicle graft was then divided longitudinally and both pedicles were swung around to the circumflex area of the heart. These grafts were sutured laterally and posteriorly to the parietal pericardium (Fig. 4). The intercostal bundles and the medial margin of the pectoral muscle were carried beneath the sternum and attached to the parietal pericardium. These structures carried the internal mammary artery onto the surface of the heart. The lateral margin of the pectoral muscle was then inverted so that the incised surfaces were in contact with the heart (Fig. 5). The fascia of the pectoral muscle was sutured; the skin was closed. The wound was not drained.

Three and one-half months have elapsed since the operation (Fig. 6). The patient has been kept in the hospital during this period but during the last six weeks he has been doing light work, such as serving trays, moving beds. *etc.* For several weeks after operation he had indigestion after meals. This has disappeared. He claims that he has no precordial pain; that the feeling of oppression over the heart has disappeared, that he has no sharp radiating pains to the shoulder and arm. He claims that he is well. He can exercise without pain, although up to the present time he has done no hard physical work. I have emphasized the importance of describing accurately what symptoms he has, but to me and to everyone else who talks with him he claims that he is better. Objectively I can say that he appears to be better. The worried expression has left him, and he has a fine spirit.

SUMMARY

The heart can be given a new blood supply experimentally. On the basis of this work a collateral vascular bed to the heart should offer some benefit to patients suffering from coronary sclerosis. A collateral vascular bed was given to a patient with coronary sclerosis on February 13, 1935. Three and one-half months after operation the patient claims that he has been greatly benefited. However, it will be necessary to have a number of such results before we can attach any clinical significance to this operation. I want to emphasize the point that the work is still in the experimental stage and I do not recommend the performance of this operation until it is established by operation upon a number of patients.

ADDENDUM: September, 1935. Seven months have now elapsed since the operation on the first patient. He continues to work as a gardener; he has no pain and he claims that he is cured. I have carried out the operation on five additional patients, making the total number seven. The third patient had an extremely marked degree of arteriosclerosis. At the operating table I could palpate the ramus descendens. It was hard, tortuous and several millimeters in diameter. The patient had been incapacitated sine 1942. One and one-half years ago a total thyroidectomy was done. He has kept his basal metabolic rate at about minus 20 by taking thyroid extract. His life had been of a vegetative nature,

spending as he did 20 hours a day in bed. Two and one-half months have elapsed since we did the cardiac anastomosis. He states that he is completely free from all pain and discomfort. He is able to take twice the dose of thyroid extract that he took before the operation and his basal metabolic rate is plus 2. He is up and about six to eight hours a day and his interest in life has returned. He is still an invalid and he is still weak, but the complete absence of pain, his normal basal metabolic rate, his increased activities and interests, are facts that may be of real significance in establishing the operation as a beneficial procedure. The fourth patient is a well known surgeon from Ashland, Kentucky, who came to me because he believed in the soundness of my operation. He had suffered a myocardial infarct with dilatation and failure. He also had diabetes mellitus. He believes that he has shown some improvement but the pulse rate on occasions becomes rapid and he has had several attacks of anginal pain since operation. A sufficiently long interval has not elapsed to comment on the other cases. These cases will be reported by Dr. Harold Feil and myself.

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DISCUSSION.—DR. JOHN B. FLICK (Philadelphia, Pa.).—The experimental work of Claude Beck, in establishing a collateral vascular bed in the myocardium of animals and thus protecting the myocardium from anoxemia due to coronary occlusion, furnishes an entirely new avenue of approach in combating cardiac ischemia due to coronary artery disease in human beings.

Sir Thomas Lewis says, "The malady originally described by Heberden under the term *angina pectoris* is one in which pain of characteristic type occurs during effort. It is by far the commonest form of malady in which angina pain occurs. The pain may be interpreted as resulting when the blood supply to the heart, or part of the heart, is limited and consequently inadequate when the heart is called upon to do work at a certain increased rate—a condition of relative ischemia."

The operative treatment of angina pectoris first proposed by Francois Frank in 1899 and first practiced by Jonnesco in 1916 was purely symptomatic and was not intended to eradicate the disease. The surgical act aimed at division of the nervous arc thus preventing the patient from recognizing the symptom pain (Elliott C. Cutler). Since then various neurosurgical procedures along similar lines have been devised for the relief of cardiac

pain and practiced with more or less success, but they have not gained greatly in popularity, possibly because of technical difficulties and possibly because of the uncertainty of the results.

Removal of the normal thyroid gland in the treatment of heart disease was proposed by Blumgart, Lavine and Berlin and was practiced for the first time on December 14, 1932, at the Peter Bent Brigham Hospital in Boston. Strikingly beneficial results from this operation have since been obtained in the group of patients suffering from angina pectoris. The rationale of this procedure for the treatment of heart disease has its basis in the reduction of the metabolic demand on the heart by the total ablation of the thyroid gland.

Beck's operation strikes directly at the myocardial ischemia and theoretically should be beneficial where the ischemia is due to organic changes in the walls of the cardiac vessels. Experience alone can determine the place in surgery of Beck's ingenious and logical operation and he is to be congratulated upon a fine piece of work which at least has proved that a collateral circulation can be established experimentally in the myocardium of animals.

DR. ELLIOTT C. CUTLER (Boston, Mass.).—I should like to be among those who congratulate Doctor Beck upon what I know to be the end-result of a vast amount of work and a most ingenious investigation.

This morning our President spoke of our job as one of keeping Nature from taking us away before our time, but I wonder whether we should apply our energies to people as young as forty, as suggested by Doctor Cheever. I had the opportunity to see Doctor Beck's first patient as he was recovering from the operation and the ordeal did not seem unusual.

It is certain that the attack proposed by Doctor Beck and the physiologic background for this procedure are much more admirable, much better suited and far more certain to hold hope for ultimate good than any procedure yet proposed for those cardiac disorders that have as beginnings and cause ischemia of the myocardium.

Whether one is to turn in muscle flaps which many of us have tried in other conditions without much success, whether one is to use the fat about the pericardium, or whatever the actual method of establishing the collateral circulation is to be, it is certain that the understanding of what seems necessary and which Doctor Beck has attempted to carry out in these experiments constitute the most logical attempt that has ever been suggested.

The difficulty, of course, is going to be the same as we all have found in other conditions, now that ingenuity of the surgeon has brought us so far apace, *i.e.*, to convince our medical colleagues that our undertakings are desirable, or perhaps educate them to help us choose which case is desirable for the definite procedure.

Knowing how in this country surgery of the pericardium has lagged behind such surgery elsewhere, partly because of the attitude of our internists, one can imagine the difficulties in this field of choosing the proper case of arteriosclerotic heart disease, angina or hypertensive heart disease, as the one best suited for such procedure. I have no doubt if Doctor Beck keeps at it, and he fortunately has a group of medical colleagues who are interested and will help him, he will be able to tell us in a few years which form of heart disease is best suited and most likely to be relieved by this procedure.

I think we should all congratulate him sincerely for an admirable piece of work.

DR. W. F. RIENHOFF, JR. (Baltimore, Md.).—Independently and without any knowledge of Doctor Beck's experiments, or interest in this problem, we

began in October, 1934, investigating different methods of supporting an interrupted coronary circulation. We have used a series of old dogs for the reason that the independent development of a collateral circulation in younger animals is so readily achieved that recovery from ligation of the right or left anterior descending coronary artery may be clinically uneventful and easily survived by the animal. Doctor Beck did not state whether his series of dogs were young or old. In the older dogs, in our experience, ligation of the left anterior descending branch of the left coronary artery almost always resulted in either ventricular fibrillation or a marked arrhythmia. Ligation of the left coronary artery before departure of the circumflex resulted invariably in ventricular fibrillation. In a small series of nine dogs, Dr. E. Cowles Andrus, Dr. August Jonas and myself obtained electrocardiographic studies before beginning the experiment, and used only those animals in whom the electrocardiograms were normal. Under ether anesthesia, an incision was made in the fifth interspace on the left side exposing the pericardial sac which latter was opened. The central tendon of the diaphragm was then incised and the omentum drawn up into the left thoracic cavity. The epicardium was then moistened with half strength iodine and the omentum wrapped, so to speak, completely about the heart, entirely covering the anterior surface. To insure maintaining this position the omentum was fastened to the epicardium by two interrupted fine silk sutures. The pericardial sac was not closed but the hiatus in the central tendon of the diaphragm was pulled snug about the omentum. After this cardio-omentopexy had been performed, Doctor Andrus found the type of electrocardiographic curve present, suggested a coronary occlusion. Whether this alteration in the electrical reaction of the dog's heart was due to the small amount of iodine painted on the epicardium or whether it was due to the two stitches placed in the heart wall, away from the coronary vessels, was not determined. Three to six weeks later, ligation of the entire left coronary artery just below its origin from the aorta and above the division into the left anterior descending and circumflex arteries was accomplished by taking a deep bite with a French No. 2 needle in the ventricular wall in the region of the left coronary artery just beneath the tip of the left auricle. The distension of the coronary veins distal to the ligature proved its location. No disturbance in cardiac rhythm followed these ligations after previously performed cardio-omentopexy, either clinically or in the electrocardiogram. The dogs were not at all ill and were up and around their cages the next day. Whereas before similar ligation was invariably fatal, not one animal succumbed after adhesion of the omentum to the heart had been produced. At the second operation extensive adhesions were revealed to have occurred between the omentum and the heart wall. These bled profusely when slightly separated. The electrocardiograph remained the same following the first and second operative procedures. In other words, ligation of the entire left coronary vessel in this series of old dogs was relatively inconsequential when the coronary circulation was properly supported by an outside source of blood supply. We are in the process of sacrificing and injecting our specimens. Carmine gelatin was used for the coronary circulation, being injected in the aorta. This solution will not go beyond the arterioles and thus will not fill the capillary bed in the myocardium. No injection mass entered the cavities of the heart and therefore the thebesian vessels were not injected. Prussian blue mass was injected into the celiac axis, thus filling the omental vessels. This mass will fill the capillary bed and in the as yet incompletely cleared specimens, seems to have penetrated the capillary branches of the coronary system.

These specimens will be reported on later. We have not entertained the hope that this procedure of cardio-omentopexy will be clinically applicable except possibly in such cases of coronary thrombosis that might be seen at an early stage of the interruption of the coronary circulation.

DR. J. SHELTON HORSLEY (Richmond, Va.).—Is there not some difference between the circulation of the blood from the omentum and that from the pectoral muscle? According to the procedure described by Doctor Beck the pectoral muscles were severed near their insertion, and a portion of the muscle turned onto the heart. This would, of course, involve destroying the nerve supply to the pectoral muscles, with consequent atrophy of the muscle and diminution of its blood supply, even if no vessels had been actually injured when the muscle was divided. The omentum, however, when brought up to the heart would suffer no trophic changes, and consequently circulation would not be impaired by such conditions as beset the pectoral muscles when they are divided near their insertion.

DOCTOR BECK.—The second patient upon whom we carried out our operation died one week later. A thrombus developed at the bifurcation of the aorta and occluded the left common iliac artery. The ischemia of the leg was very painful and the patient died within several hours. The thrombus developed at the site of an atheromatous ulcer of the abdominal aorta. The coronary arteries showed extensive and marked sclerosis. The right coronary artery was completely occluded about 14 Mm. from its ostium. The lumina of the ramus descendens and of the ramus circumflexus of the left coronary artery were markedly constricted but not completely occluded. The myocardium of the right ventricle showed extensive replacement by fat. There was no evidence of infarction in the left ventricle. The pedicle grafts of skeletal muscle and the adjacent fat and pericardium were adherent to the myocardium and for the present at least I am satisfied with this part of the operation.

In our experience ligation of the ramus circumflexus of the left coronary artery in one stage had a high mortality, even though a collateral vascular bed had been provided for the heart. Ligation in two stages is usually successful. This statement applies to the right and to either major branch of the left coronary artery.

The pedicle grafts of skeletal muscle brought in from the chest wall are deprived of some of their normal blood supply. We know, however, that the body has a great capacity to develop blood vessels and in our experiments we obtained excellent anastomoses between the vessels of cardiac muscle and of skeletal muscle. We used omentum in a number of experiments. Anastomoses between the coronary bed and the vessels of the omentum can be obtained. I am doubtful whether omentum could be used on a human patient because the opening in the diaphragm complicates the operative procedure. Experience may alter this point of view.